[*December* 7, 1945]

# Vestibular Injuries

By TERENCE CAWTHORNE, F.R.C.S.

## INTRODUCTION

THIS paper is based upon a study of the effects of deliberately destroying the end-organ of the vestibular nerve in the labyrinth for the relief of Ménière's disease in 120 cases and upon the detailed otological examination of a series of 58 closed head injuries whose convalescence was protracted mainly on account of disturbed balance.

In both groups, once the immediate effects of the injury had been overcome and the patients were ambulant, vertigo, easily provoked by sudden head movements, was the predominating symptom; whilst the most constant, and in many the sole physical sign, was an abnormal response to caloric stimulation of the labyrinth. Moreover, it was found that all these cases required the same management to aid in restoring balance and thus encourage their return to normal.

The striking similarity between the two groups, both as regards clinical features and management, led us to consider that the injury was probably in the same place in some cases, though of course it varied both in mode of production and in extent.

It is certain that in the case of labyrinthectomy it is the end-organ of the vestibular apparatus which is damaged, but as yet there is no pathological evidence available to show end-organ damage in cases of vertigo following head injury. Nevertheless the close clinical relationship between the two groups of cases leads one to believe that in both it may be the end-organ in the labyrinth that is the seat of the damage and this belief is further strengthened by the similarity of the physical signs.

The association of vestibular damage with head injuries was noted by S. H. Mygind (1918) who found that 24% of the cases he had examined had genuine vestibular symptoms. Brunner (1928 and 1940) stressed the importance of noting any nystagmus, particularly on altering the position of the head. Barre and Greiner (1932) found vertigo in 54 out of 100 cases of head injury though otological tests did not reveal any correspondence between cochlear and vestibular changes. Linthicum and Rand (1931) urged that post-concussional vertigo should not be dismissed without detailed neuro-otological tests, and Voss (1934) suggested that the term "labyrinthine concussion" might be used for these cases. Glaser (1937) found vestibular abnormalities in 76% of 66 cases, most of which he attributed to central changes. Barmoel and Marks (1941) stressed the value of a full otological examination in all cases of head injury followed by vertigo and mentioned the importance in the caloric test of using both the hot and cold stimulus to bring out any latent tendency to nystagmus.

In the general descriptions of concussion the frequency of vertigo as a sequel of head injury was particularly noted by Osnato and Giliberti (1927) and by Symonds (1937 and 1943) and Russell (1932), all of whom appreciated the possibility of vestibular disturbance in such cases.

## MODE OF INJURY

(*a*) *Labyrinthectomy* (Cawthorne, 1943).—The site and mode of injury in the cases of labyrinthectomy that we have studied are quite clear. After opening into the perilymph space the membranous external canal is seized and removed. This has always resulted in a total loss of vestibular as well as cochlear function. We have found that the same result follows merely opening the endolymph space by tearing the membranous canal across or by coagulating the membranous canal with diathermy.

(*b*) *Labyrinthotomy.*—Another form of deliberate operative injury to the labyrinth that I think deserves mention is the opening of the perilymph space for the relief of deafness. Here the injury to the end-organ should be slight because an essential precaution of the operation is to avoid damage to the membranous labyrinth. Nevertheless, even when one can be reasonably certain that the membranous labyrinth has not been touched the disturbance produced is instantaneous and considerable and must be due to altered physical conditions rather than to direct injury or inflammatory reaction. This aspect of labyrinthotomy requires careful consideration because it has been my experience that in such cases vertigo forms a prominent and sometimes protracted feature of the post-operative course and prospective candidates for this operation should be warned

beforehand that they may be troubled by vertigo afterwards They are, of course, helped by the same management as the other cases under consideration.

(c) *Post-concussion.*—Certain experimental work has been done to show that head injuries may be accompanied by changes both in the vestibular end-organ and its connexions in the central nervous system. Brunner (1928, 1940) found that blows on the fixed head caused changes in the peri- and endo-lymphatic spaces and also certain changes, mainly vascular, in the brain-stem as well as in other parts of the central nervous system. More recent work by Denny-Brown and Russell (1941) has revealed that the clinical picture of concussion could be easily produced experimentally by blows from a hammer with a striking speed of up to 40 feet per second upon the occipito-parietal region of animals whose heads were free to move with the impact of the blow. They termed this "Acceleration Concussion". Similar injuries to a fixed head they termed "Compression Concussion". A much smaller striking force was required to produce acceleration concussion than compression concussion and was attributed to a direct physical injury to the neurons which was reversible. It was obtained in the decerebrate animal both with and without section of the 8th nerves.

Nearly all the concussion cases with prolonged vertigo that we have observed were the result of acceleration concussion and I think that there is a very good reason why this is so.

As both the cochlear and vestibular end-organs rely for their essential stimulus upon displacement or deformation by a movement of endolymph, it is reasonable to suppose that if one end-organ suffers actual damage from over-stimulation by excessive endolymph movement, then over-stimulation may in a like manner cause damage to the other end-organ.

In the case of the cochlea, it is known that exposure to loud sounds (e.g. gunfire, boiler-making) may result in a disturbance of the cochlear fluids that is sufficient to cause permanent damage to the organ of Corti.

In the vestibular part of the labyrinth the end-organs (maculæ of the utricle and cupolæ of the canals) are so designed as to be exposed to pressure or traction by displacement of the endolymph in response to acceleration and deceleration movements of the head. As the maculæ and cupolæ are stimulated by the slightest and gentlest movements of the head, it is reasonable to assume that they would be exposed to a heavy strain when stimulated by the sudden and violent jerking of the head that is part of acceleration concussion. It might, therefore, be expected that the vestibular end-organ in the labyrinth would be particularly vulnerable to injury in concussion. The facts that vertigo is the cardinal symptom of a damaged labyrinth and that a frequent sequel of concussion is vertigo support this hypothesis and I hope that it may be possible at a later date to produce more concrete evidence to show that the vestibular end-organ is not infrequently injured in acceleration concussion.

## Clinical Features

The symptoms and signs that follow immediately upon an injury to the labyrinth are widespread and are often so terrifying in their intensity that observers unused to the ways of the labyrinth may find it difficult to believe that such a profound disturbance can be caused by injury to such a modest organ.

The overwhelming vertigo, the awful sickness and the turbulent eye movements—all enhanced by the slightest movement of the head—combine to form a picture of helpless misery that has few parallels in the whole field of injury and disease.

This lurid picture is, of course, seen when a previously active labyrinth is completely and suddenly overwhelmed and might conveniently be known as the syndrome of acute vestibular failure. The intensity of the symptoms will, however, vary from case to case, depending upon the nature and extent of the injury and they may sometimes be masked by the effects of injuries elsewhere. Fortunately the body is soon able to adjust itself to altered conditions even if normal balance between the two labyrinths is not quickly restored, and within a few days of injury the acute phase dies down, leaving a residue of effects which deserve our very careful attention. This residue is seen after operations on the labyrinth and in the so-called post-concussion syndrome where vertigo in one form or another plays a prominent part. The vertigo, giddiness or dizziness consists of a sensation of apparent movement, either of the subject in relationship to his surroundings or of the surroundings in relationship to the subject. The direction and extent of the apparent movement are immaterial. The essential feature is a sensation of movement that does not, in fact, take place. It is typically provoked by sudden alteration of

posture or by sudden turning movements of the head. In most cases it is momentary, but in the postural giddiness induced when the head moves forwards or backwards, the sensation may remain for some time, though if the patient can be persuaded to maintain the offending position, the symptom gradually dies down, usually within a minute. In such cases there may be an accompanying nystagmus lasting for about the same time as the vertigo.

In the momentary vertigo following sudden head turning it is not often possible to observe any involuntary eye movements, though sometimes a fleeting flicker may be present. This is the common picture following labyrinth operations and many concussional head injuries. In another group of head injury cases the complaint may be one of continuous slight movement of the ground as though on a slightly rolling ship.

Frequently after labyrinthectomy and sometimes in the post-concussion cases there is a complaint of inability to focus the gaze upon an object for any length of time and of a dislike of looking at quickly moving objects. This is often sufficient to demand an ophthalmic examination which, however, rarely reveals any significant change.

Instability in the dark and on moving up and down stairs or on an inclined plane is usual and, as you will hear later, is catered for in the treatment. A tendency to tire quickly on physical exertion is almost the rule, particularly after labyrinthectomy, and it has been found advisable to warn patients that their return to normal life may have to be gradual and will depend largely upon their ability to overcome this fatigue.

If all these symptoms are not given careful attention and particularly if any adequate explanation is not forthcoming for them, a sense of insecurity may be engendered which will encourage the patient, particularly if he is of an unstable temperament, to drift into a state of chronic invalidism sometimes amounting almost to helplessness.

Headache, which is such a common sequel of concussion, is usually not seen after operation on the labyrinth and cannot, I think, be considered as part of a vestibular disorder, though it may be a most distressing and intractable feature of the post-concussion state. In long-standing cases there is sometimes an excessive and even bizarre disturbance of balance that is clearly out of proportion to the physical signs.

Out of the 58 cases of head injury which were submitted to a full otological examination 56 showed deviations from the normal in their response to caloric stimulation of the labyrinth according to the technique first described before this Section by Hallpike in 1942.

TABLE I

| | | |
|---|---|---|
| Total cases | | 58 |
| Abnormal caloric response | | 56 |
| | Directional preponderance | 39 |
| | Canal paresis | 17 |
| Normal caloric response | | 2 |

and more fully elaborated by Fitzgerald and Hallpike (1942) and Cawthorne, Fitzgerald and Hallpike (1942) (Table I). Of the remaining two cases, in one the vertigo had disappeared some days before the caloric test and in the other a transient dizziness on bending down had been experienced ever since a head injury two years previously. Of the 56 cases who showed an abnormal vestibular response only 24 had damage to the cochlea (Table II).

TABLE II

| | | |
|---|---|---|
| Normal hearing | | 34 |
| | Directional preponderance | 23 |
| | Canal paresis | 9 |
| | Normal calorics | 2 |
| Abnormal hearing | | 24 |
| | Directional preponderance | 16 |
| | Canal paresis | 8 |

These findings strengthen my belief that in persistent vertigo following concussion the vestibular end-organ is a likely seat of the damage. I would like to emphasize that signs of such damage can only be demonstrated by a careful and detailed otological examination.

## Treatment

The impression that we have gained from our observation of these cases during the past eight years is that the disturbance of balance caused by an injury to the vestibular apparatus has a profound effect on the well-being of the patient. This is not surprising when we recall that the sense of balance is one of the most primitive faculties with which we are equipped, appearing in the biological scale before the senses of sight and hearing

and possibly before the senses of taste and smell, though of course it has not as yet been elevated to the dignity of a "sense".

To the more impressionable patients a labyrinthine disturbance may make them feel that the end of the world has arrived and I am told by sufferers from sea-sickness that in the acutest phase of their distress, they wish that it had.

Hence it is not surprising that the clinical picture which we have been considering is not infrequently complicated by psychological disturbances which may be sufficient to divert attention away from the underlying cause.

We have found that once the true nature of the disturbance has been established an explanation of the state of affairs, combined with graduated exercises especially designed to encourage head and eye movements, form the most satisfactory basis for hastening recovery (Cawthorne, 1945).

Since the head exercises, which Dr. Cooksey will describe, were instituted by him and carried out by Miss Swan and Miss Hudson at Horton Emergency Hospital, the rate of recovery after operations on the labyrinth has been greatly hastened and we now expect that such cases can resume their normal occupation from within a month of operation, though of course it may sometimes be advisable to recommend a change of occupation. The post-concussion cases, for whom these exercises are particularly useful can, if they are tackled reasonably soon after injury, usually be prevented from drifting into chronic invalidism. The poorest responses to exercises and rehabilitation were always seen in those cases which had been allowed to drift for months with but little planned treatment.

The management of these cases has been made easy and pleasant for me and for my patients because of the way in which it has been possible for Dr. Cooksey and his assistants, the clinicians and ward sisters, all to work together and to share the problems raised by these cases.

I shall always be grateful to Dr. Cooksey and his assistants for their help and to my colleagues at King's College Hospital, the National Hospital, Queen Square, Horton Emergency Hospital and Hurstwood Park Emergency Hospital and to friends elsewhere for referring their cases to us. We have been able to help some, we have failed with others, but I know that we have learnt a lot from all of them.

REFERENCES.

BARMOELS, and MARKS, M. I. (1941) *Arch. Otolaryng.* Chicago, **33,** 204.
BARRE, J. A., and GREINER, G. (1932) *Rev. Oto-Neuro-Ophtal.*, **10,** 633.
BRUNNER, H. (1928), in Alexander and Marburg's *Handbuch der Neurologie des Ohres*, Vol. **2**, pt. 1, p. 273.
—— (1940) *Laryngoscope*, **50,** 921.
CAWTHORNE, T. (1943) *J. Laryng.* **58,** 363.
—— (1945) *J. chart. Soc. Physiotherapy*, April.
——, FITZGERALD, G., and HALLPIKE, C. S. (1942). *Brain*, **65,** 138.
DENNY-BROWN, D., and RUSSELL, W. R. (1941) *Brain*, **64,** 93.
FITZGERALD, G., and HALLPIKE, C. S. (1942) *Brain*, **65,** 115.
GLASER, M. A., (1937) *Ann. Otol. etc.*, St. Louis, **46,** 387.
LINTHICUM, F. H., and RAND, C. W. (1931) *Arch. Otolaryng.*, Chicago, **13,** 785.
MYGIND, S. H. (1918) *Acta Otolaryng.*, Stockh., **1,** 515.
OSNATO, M., and GILIBERTI, V. (1927) *Arch. Neurol. Psychiat.*, Chicago, **18,** 181.
RUSSELL, W. R. (1932) *Brain*, **55,** 549.
SYMONDS, C. P. (1937) *Guy's Hosp. Gaz.*, **51,** 461.
—— (1943) Injuries of Skull, Brain and Spinal Cord. S. Brock, Ed., p. 65. Baltimore.
VOSS (1934) *J. Amer. med. Ass.*, **103,** 1721.

## Rehabilitation in Vestibular Injuries

By F. S. Cooksey, O.B.E., M.D.

In 1941 Mr. Cawthorne put to me the principles governing the restoration of fitness after injury to the vestibule and sought my help to develop a system of rehabilitation for these cases. It happened that about the same time I had arranged with the Staff of the Head Injury Centre at Hurstwood Park Hospital to undertake the rehabilitation of late cases of the post-concussion syndrome in my Department at Horton Emergency Hospital. Thus the system of rehabilitation which I am about to describe was developed for the post-concussion syndrome as a whole as well as for the particular problem of vestibular injury.

The symptom complex for which I had to provide consists of headache, vertigo including the so-called "black-outs", impaired mental concentration and deafness in a proportion of cases. Of these symptoms the vertigo may be the most disturbing; but, fortunately is usually amenable to treatment along the lines I shall describe.